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Perfluorochemicals in Pooled Serum Samples from United States Residents in 2001 and 2002

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Manufacturers have used perfluorochemicals (PFCs) since the 1950s in many industrial and consumer products, including protective coatings for fabrics and carpet, paper coatings, insecticide formulations, and surfactants. Some PFCs are persistent ubiquitous contaminants in the environment and in humans. Exposures to PFCs result in potential developmental and other adverse effects in animals. The sources of human exposure to PFCs and the potential health risks associated with exposure are still unclear, and differences in patterns of human exposure may vary. We measured the serum concentrations of perfluorooctane sulfonic acid (PFOS), perfluorooctanoic acid (PFOA; C8), perfluorohexane sulfonic acid (PFHxS), and 8 other PFCs in 54 pooled serum samples collected from 1832 participants of the 2001−2002 National Health and Nutrition Examination Survey. Participants were 12 years of age and older. The pools represented three major racial groups/ethnicities (non-Hispanic blacks, non-Hispanic whites, and Mexican Americans), four age categories (12−19 years, 20−39 years, 40−59 years, and 60 years and older), and both genders. PFCs were extracted from 100 μL of serum using on-line solid-phase extraction coupled to isotope dilution-high performance liquid chromatography-tandem mass spectrometry. The limits of detection ranged from 0.05 ng/mL to 0.2 ng/mL. The concentrations of most PFCs were similar among the four age groups. For PFOS, the estimated least-squares mean (LSM) concentrations among non-Hispanic white males (40.19 ng/mL) and females (23.97 ng/mL) were greater than among non-Hispanic black males (18.27 ng/mL) and females (17.93 ng/mL) or Mexican American males (13.71 ng/mL) and females (10.40 ng/mL). Similarly, for PFOA, the LSM concentrations among non-Hispanic white males (6.98 ng/mL) and females (3.97 ng/mL) were greater than among non-Hispanic black males (3.62 ng/mL) and females (2.85 ng/mL) or Mexican American males (2.89 ng/mL) and females (2.08 ng/mL). Non-Hispanic whites had also greater LSM concentrations of PFHxS than non-Hispanic blacks and Mexican Americans. These findings indicate different patterns of human exposure to PFCs among the population groups examined and stress the importance of conducting research to identify the environmental sources and pathways of human exposure to PFCs.

Introduction

Since the 1950s, perfluorochemicals (PFCs) have been widely used in commercial applications including surfactants, lubricants, paper and textile coatings, polishes, food packaging, and fire-retarding foams. Several of these PFCs, including perfluorooctane sulfonic acid (PFOS) and perfluorooctanoic acid (PFOA; C8), are persistent in the environment and humans, and have been found around the world in wildlife (*1*−*10*). Exposure to PFCs among the general population is also widespread, although the ranges of concentrations and frequencies of detection of PFCs among countries and even within the same region may vary (*10*−*20*).

The results of animal studies suggest potential developmental and other adverse effects associated with exposures to PFOS (*21*−*28*) and PFOA (*24*, *29*−*32*). In contrast, no clear association has been established between human exposure to PFCs and adverse health effects in a small number of occupational studies. Medical surveillance of fluorochemical production workers did not show substantial changes in serum cholesterol or lipoproteins or in reproductive hormones, or clinical hepatic toxicity, all of which are consistent with the known toxicologic effects of PFCs in animals (*33*−*37*). In turn, two retrospective cohort mortality studies showed increased risk of some cancers among fluorochemical production workers (*38*, *39*).

In 2002, the 3M Company—the sole manufacturer of PFOS in the United States and the principal manufacturer worldwide—discontinued the production of PFOS and related perfluorooctanyl chemistries. PFOA and its salts are still being manufactured by other companies for use primarily in the production of fluoroelastomers and fluoropolymers, such as polytetrafluoroethylene (Teflon) and polyvinylidine fluoride. These are used in the automotive, mechanical, aerospace, chemical, electrical, medical, and building/construction industries, and in consumer products (e.g., coatings on paper, textiles, and carpet; personal care products; and nonstick coatings on cookware). In 2003 and 2005, the U.S. Environmental Protection Agency (U.S. EPA) released preliminary risk assessments on PFOA, indicating potential human exposure to PFOA in the United States, and suggesting additional research on human exposure for evaluating potential health effects associated with exposures to PFOA and other PFCs (*40*).

We measured the serum concentrations of 11 PFCs in 54 pooled serum samples collected from participants of the 2001−2002 National Health and Nutrition Examination Survey (NHANES). Although the concentrations of some PFCs have been measured in children 2−12 years of age (*19*) and in adults more than 20 years old in the United States (*17*, *18*), this study provides the first estimation of mean concentrations of PFOS, PFOA, and 9 other PFCs in a sample of the U.S. population 12 years of age and older. We examined demographic differences (e.g., age, gender, race) among the pools, and compared the prevalence and magnitude of exposure to PFCs in these samples with those previously reported in the United States (*11*, *13*, *15*, *17*−*19*) and other countries (*10*−*12*, *15*, *16*, *41*, *42*).

Materials and Methods

Serum samples, used to prepare the pools analyzed for the study, were selected from those obtained by venipuncture

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TABLE 1. Number of Pooled Serum Samples Prepared[a] from NHANES 2001−2002 Participants Per Demographic Group

		age group (year)			
race-ethnicity	gender	12−19	20−39	40−59	60+
non-hispanic white	male	3 (105,102)	3 (112,102)	3 (125,102)	4 (154,136)
	female	3 (120,102)	4 (155,136)	3 (120,102)	4 (157, 136)
non-hispanic black	male	3 (115,102)	1 (54, 34)	1 (53, 34)	1 (31, 31)
	female	3 (123,102)	1 (63, 34)	1 (45, 34)	1 (44, 34)
mexican american	male	3 (108,102)	2 (67, 67)	1 (49, 34)	1 (36, 34)
	female	4 (140,136)	2 (84, 68)	1 (45, 34)	1 (45, 34)

[a] In parenthesis, number of individual serum samples available, number of individual serum samples used to prepare the pool(s).

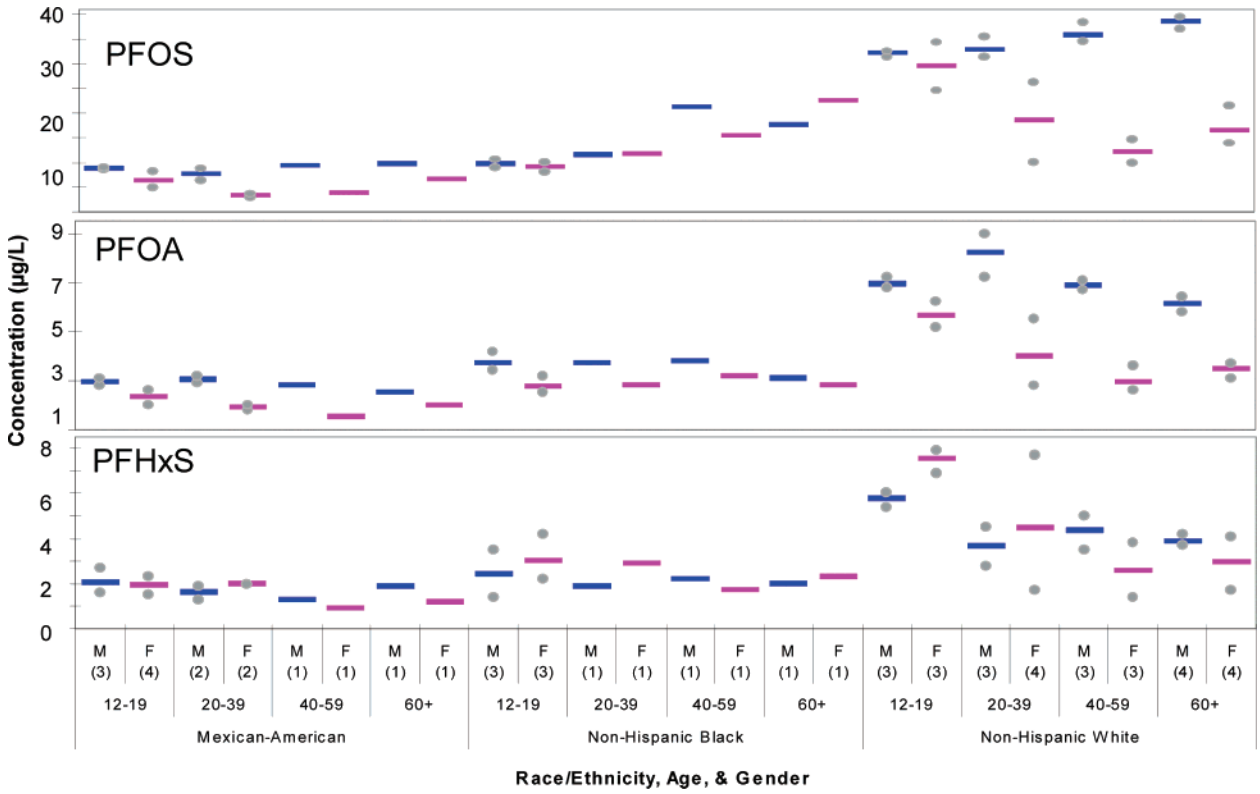

FIGURE 1. Minimum (lower filled circle), mean (bar), and maximum (upper filled circle) concentrations of PFOS, PFOA, and PFHxS in pooled serum samples collected in the United States between 2001 and 2002. These analytes were detected in all of the pools examined.

from 2150 participants, a random one-third subsample of people 12 years of age and older of NHANES 2001−2002 representative of the U.S. general population for this age range. NHANES, conducted by the National Center for Health Statistics (NCHS) of the Centers for Disease Control and Prevention (CDC), is an ongoing survey designed to measure the health and nutrition status of the civilian noninstitutionalized U.S. population (*43*). The sampling scheme for NHANES 2001−2002, a complex multistage area probability design, included 11 039 persons. Data were collected through household interviews and by standardized physical examinations conducted in mobile examination centers. On the basis of self-reported data, a composite race/ethnicity variable helped define three major racial/ethnic groups: non-Hispanic blacks, non-Hispanic whites, and Mexican Americans. Participation of the human subjects occurred only after informed consent was obtained (*43*).

After collection, serum specimens were divided into aliquots and stored cold (2−4 °C) or frozen until shipped on dry ice to CDC's National Center for Environmental Health. Serum samples were stored frozen at −20 °C until needed. The 2150 individual serum samples available were categorized in 24 demographic groups, each representing a combination of race/ethnicity, gender, and age (12−19 years, 20−39 years, 40−59 years, and 60 years and older) (Table 1). A total of 1832 individual serum samples were used to prepare the 54 pooled serum samples analyzed for this study. Based on the number of individual serum samples per demographic group, multiple pools were available for 14 groups, and one pool was available for 10 groups (Table 1). To ensure that no individual sample overly influenced the pooled results, all serum samples included in any one pool (25 mL each) were of equal volume (minimum of 750 µL). Most pools included 34 individual serum samples randomly selected. Only 31 individual serum samples were available for the pool representing non-Hispanic black men 60 years of age or older. Similarly, one of the pools representing Mexican American men between 20 and 39 years of age included 33 individual specimens. After preparation, the serum pools were stored at −20 °C until analysis.

Through a multiple reaction monitoring experiment using online solid-phase extraction coupled to reversed-phase high-performance liquid chromatography−tandem mass spectrometry (*44*), we measured the following analytes: perfluorooctane sulfonamide (PFOSA), 2-(*N*-ethyl-perfluorooctane sulfonamido) acetic acid (Et-PFOSA-AcOH), 2-(*N*-methyl-

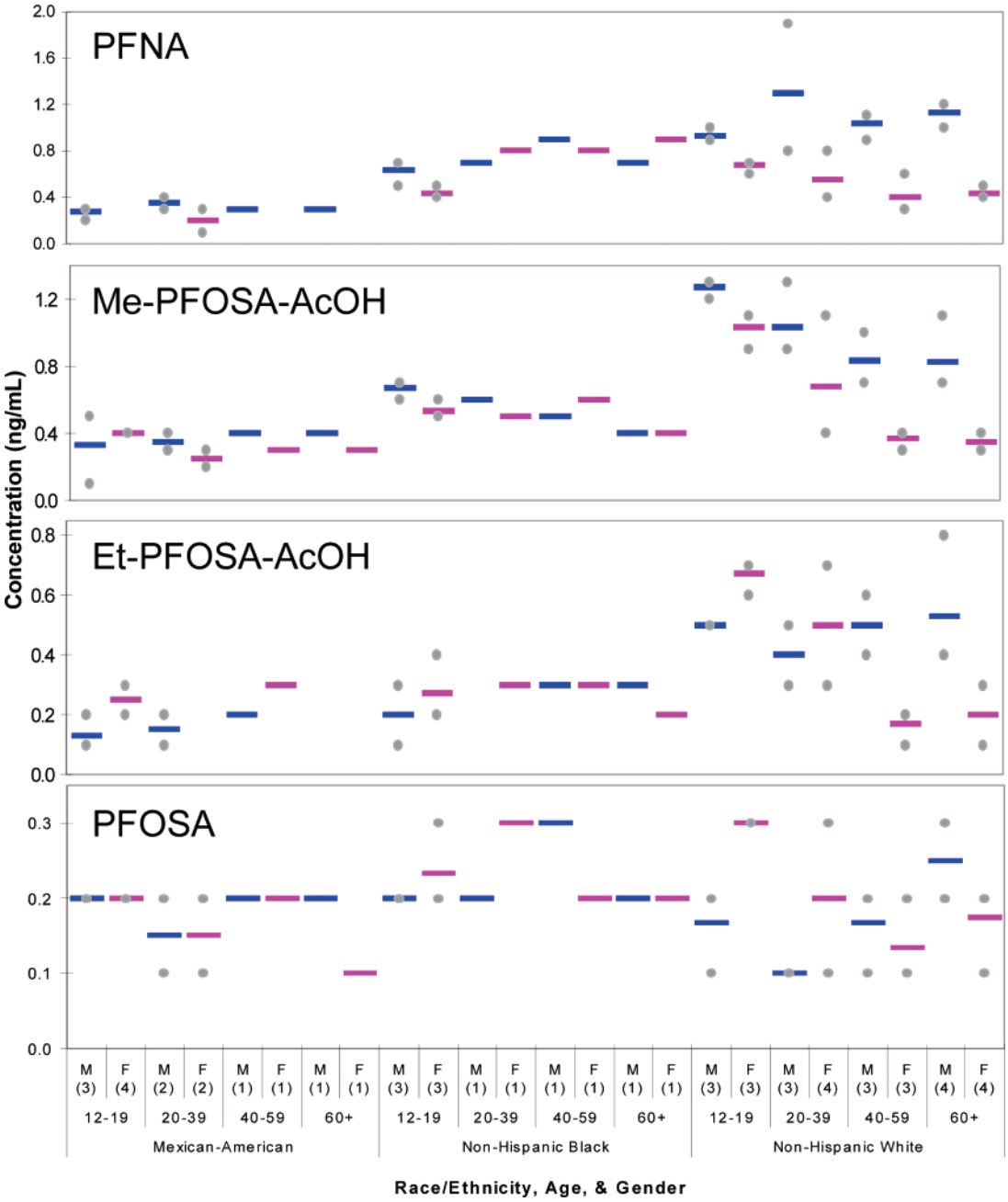

FIGURE 2. Minimum (lower filled circle), mean (bar), and maximum (upper filled circle) concentrations (frequency of detection) of PFNA (92.6%), PFOSA (100%), Me-PFOSA-AcOH (98.1%), and Et-PFOSA-AcOH (81.5%) in pooled serum samples collected in the United States between 2001 and 2002.

perfluorooctane sulfonamido) acetic acid (Me-PFOSA-AcOH), perfluorohexane sulfonic acid (PFHxS), PFOS, PFOA, perfluorohexanoic acid (PFHxA), perfluorononanoic acid (PFNA), perfluorodecanoic acid (PFDeA), perfluoroundecanoic acid (PFUA), and perfluorododecanoic acid (PFDoA). The analytical method used has been described in detail (*44*). Briefly, without protein precipitation, only dilution with 0.1 M formic acid, one aliquot of 100 μL of serum was injected into a commercial column switching system allowing for concentration of the analytes on a C18 solid-phase extraction column. This column was placed automatically in front of a C8 analytical high-performance liquid chromatography column for chromatographic separation of the analytes. Detection and quantification were done using negative-ion TurboIonSpray ionization, a variant of electrospray ionization, tandem mass spectrometry. Three isotope-labeled internal standards were used for quantification: $^{18}O_2$−PFOSA, $^{18}O_2$−PFOS, and $^{13}C_2$−PFOA. To compensate for the lack of isotope-labeled internal standards for the other analytes and account for matrix effects, the calibration standards were spiked into calf serum. Spiked serum was analyzed repeatedly to determine the limit of detection (LOD), accuracy, and precision of the method. LOD was calculated as $3S_0$, where S_0 is the standard deviation as the concentration approaches zero (*45*). LOD was 0.2 nanograms per milliliter (ng/mL), except for PFHxS (0.1 ng/mL) and PFOSA (0.05 ng/mL). The standard accuracies (77−109%) and their relative standard deviations (5−24%) were obtained at three spike levels (LOD, 1.25 ng/mL and 12.5 ng/mL) (*44*). To correct for the endogenous PFOS present in the calf serum, we increased the calculated PFOS concentrations by 0.6 ng/mL. No corrections were applied to the other PFC concentrations (*44*).

Quality control (QC) materials were analyzed with the pooled samples to ensure the accuracy and reliability of the data. Low-concentration (QCL; ~3 ng/mL to ~9 ng/mL, depending upon the analyte) and high-concentration (QCH; ~10 ng/mL to ~30 ng/mL, depending upon the analyte) QC materials were prepared from a base calf serum pool, dispensed in 3-mL aliquots and stored at −20 °C. We characterized each QC material through repeated measurements spanning at least 3 weeks, to define the mean concentrations and the 95 and 99% control limits of PFCs. The coefficients of variation of 30 repeated measurements for each serum pool ranged between 6 and 16% for all analytes (*44*). The analytical batch consisted of nine calibration standards and 61 samples (including two QCH, two QCL, two reagent blanks, and one serum blank). We evaluated the concentrations of the two QCH and the two QCL, averaged to obtain one measurement of QCH and of QCL per batch, using standard statistical probability rules.

We analyzed the data using Statistical Analysis System (SAS Institute, Cary, NC) software, version 9.1. Results less than the LOD were imputed a value of the LOD divided by the square root of 2 for calculations (*46*). Because most pools consist of approximately 34 samples, the measured value of each one pool is the arithmetic average of the individual samples making up the pool. The concentrations of environmental chemicals in individual samples from the U.S. population generally tend to be log-normally distributed; therefore, the measured values for the pools are comparable to arithmetic averages of log-normal results. As arithmetic averages, pool measured concentrations should be approximately Gaussian; thus, we chose to use unbalanced analysis of variance (ANOVA) methods.

Because the samples constituting the pools originated from NHANES 2001−2002, which was designed to be representative of the noninstitutionalized U.S. population, the pooled results do provide good coverage of the U.S. population. By pooling across design cells, however, we cannot be assured that estimates based on the pooled samples are unbiased. In addition, least squares means (LSM) from the ANOVA represent positively biased estimates of the central values of the 24 demographic groups. This positive bias arises from the inherent bias associated with each pool measured result, which is comparable to an arithmetic average of log-normal results. No attempt was made at this time to correct for this bias.

In our initial unbalanced ANOVA models, we explored the main effects of age, gender, and race/ethnicity, and we included all possible two-way interactions between these effects. Since the measured values of the pools do not reflect the survey variability among samples within pools, we adjusted the calculated mean square errors from the ANOVAs by assuming a specified design effect. We did not know the design effect associated with these estimates, but the NCHS of the CDC has estimated the average design effect from previous surveys to be near 1.5 and to rarely be less than 1.0 (*47*). Therefore, we chose 3.5 as a conservative estimate of how large the design effect might be and adjusted all significance values and standard errors accordingly. Using these adjusted significance values, we considered analyses to be statistically significant when $p \leq 0.05$, and nominally or marginally significant when $0.05 < p < 0.1$. Because of the potential for bias in the estimates and the large number of statistical tests at the 0.05 level, any differences between subgroups should only be considered valid for exploratory purposes. Results are presented in ng/mL.

Results

Mean, maximum and minimum concentrations of PFOS, PFOA, PFHxS, PFNA, PFOSA, Me-PFOSA-AcOH, and Et-PFOSA-AcOH for the 24 possible combinations of race/ethnicity, gender, and age groups are shown in Figures 1 and 2. PFOS was found at the highest concentrations, followed by PFOA and PFHxS. PFOS, PFOA, PFHxS, and PFOSA were detected in all of the pools analyzed, while other analytes were detected less frequently: Et-PFOSA-AcOH (81.5%), Me-PFOSA-AcOH (98.1%), and PFNA (92.6%). PFHxA, PFDeA, PFUA, and PFDoA were infrequently detected (<30% of the pools examined), and will not be discussed further.

TABLE 2. Significance Levels Associated with Statistically Significant Factors from an Unbalanced ANOVA after Adjustment of the Mean Square Error to Account for an Assumed Design Effect of 3.5

analyte[a]	source	probF[b]	probF_adj
PFOS	race	<0.0001	<0.0001
	gender	<0.0001	0.0205
	gender*race	<0.0001	0.0294
PFOA	race	<0.0001	<0.0001
	gender	<0.0001	0.001
	gender*race	<0.0001	0.0335
PFHxS	race	<0.0001	0.0063
PFNA	race	<0.0001	<0.0001
	gender	<0.0001	0.0072
	gender*race	0.0001	0.0502
Et-PFOSA-AcOH	race	<0.0001	0.0143
Me-PFOSA-AcOH	race	<0.0001	0.0218

[a] Perfluorooctane sulfonic acid; PFOA, perfluorooctanoic acid; PFHxS, perfluorohexane sulfonic acid; Et-PFOSA-AcOH, 2-(*N*-ethyl-perfluorooctane sulfonamido) acetic acid; Me-PFOSA-AcOH, 2-(*N*-methyl-perfluorooctane sulfonamido) acetic acid; PFNA, perfluorononanoic acid. Results less than the limit of detection (LOD) were imputed a value of the LOD divided by the square root of two for calculations. [b] ProbF is the observed significance level from an unbalanced ANOVA assuming the measured pool values are independently identically distributed, which is unlikely since the original sample data were obtained from a stratified multistage probability design. Therefore, adjusted significance levels (probF_adj) were also computed assuming a design effect equal to 3.5, chosen as a conservative estimate of how large the design effect might be. Only factors with probF_adj less than 0.05 were included in the final ANOVA models.

For PFOS and PFOA, the sex by race interaction was significant (Table 2). However, the main effect of age was not. For PFOS, the concentration difference between males and females was only significant ($p < 0.0001$) for non-Hispanic whites. Among males, PFOS concentrations for non-Hispanic whites were statistically different from those of Mexican American ($p < 0.0001$) and non-Hispanic blacks ($p < 0.0001$). Among females, the concentrations of PFOS for Mexican Americans and non-Hispanic whites were statistically different ($p = 0.0025$). For PFHxS, Et-PFOSA-AcOH and Me-PFOSA-AcOH, there were no significant interactions in the design effect adjusted model, and race was the only significant effect (Table 2). Serum concentrations among Mexican Americans did not differ from those among non-Hispanic Blacks for PFHxS ($p = 0.4877$), Et-PFOSA-AcOH ($p = 0.5300$), and Me-PFOSA-AcOH ($p = 0.2718$). However, PFHxS concentrations among non-Hispanic whites were significantly greater than among Mexican American ($p = 0.0028$) and non-Hispanic blacks ($p = 0.0375$). Compared to Mexican Americans, non-Hispanic whites had greater concentrations of Et-PFOSA-AcOH ($p = 0.0063$) and Me-PFOSA-AcOH ($p = 0.0066$). Although Et-PFOSA-AcOH and Me-PFOSA-AcOH concentrations were greater for non-Hispanic whites than for non-Hispanic blacks, the differences were not statistically significant ($p = 0.0578$, Et-PFOSA-AcOH; $p = 0.1708$, Me-PFOSA-AcOH). For PFNA, the gender by race interaction was right at significance ($p = 0.0502$) in the model adjusted for design effects (Table 2). The PFNA concentrations among males and females were only significantly different for non-Hispanic whites ($p < 0.0001$). Among males, Non-Hispanic whites had PFNA concentrations significantly

TABLE 3. Least Squares Means Estimates of Serum Concentrations and Their corresponding 95% confidence limits

analyte[a]	gender	race	mean concentration (ng/mL)	L95[b] (ng/mL)	U95 (ng/mL)	L95_adj[c] (ng/mL)	U95_adj (ng/mL)
PFOS	female	MA	10.40	6.76	14.04	3.59	17.21
	female	NHB	17.93	13.73	22.14	10.07	25.80
	female	NHW	23.97	21.22	26.72	18.82	29.12
	male	MA	13.71	9.82	17.61	6.43	20.99
	male	NHB	18.27	14.06	22.47	10.40	26.13
	male	NHW	40.19	37.34	43.05	34.85	45.53
PFOA	female	MA	2.08	1.50	2.65	1.01	3.14
	female	NHB	2.85	2.19	3.51	1.61	4.09
	female	NHW	3.97	3.54	4.40	3.16	4.78
	male	MA	2.89	2.27	3.50	1.74	4.03
	male	NHB	3.62	2.96	4.28	2.38	4.85
	male	NHW	6.98	6.53	7.43	6.14	7.82
PFHxS		MA	1.75	1.05	2.46	0.44	3.07
		NHB	2.44	1.65	3.23	0.97	3.92
		NHW	4.33	3.80	4.85	3.34	5.31
PFNA	female	MA	0.13	−0.01	0.26	−0.12	0.37
	female	NHB	0.63	0.48	0.78	0.35	0.92
	female	NHW	0.51	0.41	0.61	0.32	0.69
	male	MA	0.30	0.16	0.44	0.04	0.56
	Male	NHB	0.70	0.55	0.85	0.42	0.98
	Male	NHW	1.10	1.00	1.20	0.91	1.29
Et-PFOSA-AcOH		MA	0.17	0.10	0.25	0.03	0.32
		NHB	0.24	0.16	0.33	0.08	0.40
		NHW	0.43	0.37	0.49	0.32	0.54
Me-PFOSA-AcOH		MA	0.35	0.22	0.48	0.10	0.59
		NHB	0.55	0.40	0.70	0.28	0.82
		NHW	0.78	0.68	0.88	0.60	0.96
PFOSA			0.19	0.18	0.21	0.16	0.23

[a] PFOS, perfluorooctane sulfonic acid; PFOA, perfluorooctanoic acid; PFHxS, perfluorohexane sulfonic acid; PFOSA, perfluorooctane sulfonamide; Et-PFOSA-AcOH, 2-(*N*-ethyl-perfluorooctane sulfonamido) acetic acid; Me-PFOSA-AcOH, 2-(*N*-methyl-perfluorooctane sulfonamido) acetic acid; PFNA, perfluorononanoic acid, MA, Mexican Americans; NHB, non-Hispanic blacks; NHW, non-Hispanic whites. [b] L95 and U95 are the lower and upper 95% confidence limits, respectively, assuming the measured pool values are independently identically distributed. [c] L95_adj and U95_adj are the 95% confidence limits, respectively, that result when assuming a design effect of 3.5.

different from Mexican Americans ($p < 0.0001$) and non-Hispanic blacks ($p = 0.0224$); PFNA concentrations for Mexican Americans and non-Hispanic blacks were also statistically different ($p = 0.0416$). For females, PFNA concentrations among Mexican Americans differed from non-Hispanic whites ($p = 0.0155$) and non-Hispanic blacks ($p = 0.0086$), but non-Hispanic blacks and non-Hispanic whites had concentrations comparable to one another ($p = 0.4551$). For PFOSA, without adjustment for design effects, the sex by age interaction was significant ($p = 0.0078$). However, when adjusting for design effects, no interactions or main effects were significant.

The estimated least squares mean (LSM) concentrations of PFCs for selected demographic groups are given in Table 3.

Discussion

PFOS, PFOA, and PFHxS were detected among all ages and racial/ethnic groups, and both genders from a sample of the U.S. general population 12 years of age and older (Figure 1). Other PFCs (PFOSA, Et-PFOSA-AcOH, Me-PFOSA-AcOH, and PFNA) were detected in more than three-quarters of the samples examined (Figure 2). These findings confirm that exposure to PFOS, PFOA, PFHxS, and other PFCs is widespread in the United States.

In agreement with previous studies (*13*−*15*, *17*−*19*), PFOS was present at the highest concentrations, followed by PFOA. The mean concentrations of PFOS and PFOA were greater than those of PFHxS and other PFCs measured. The concentration order and concentrations of PFOS, PFOA, and PFHxS are comparable with those reported previously for adults (*17*), the elderly (*18*), and children (*19*) in the United States. Furthermore, the concentration ranges of PFOS were similar to those found in selected populations in other countries, including Canada (*41*), Japan (*10*, *16*), and in Europe (*15*).

Despite the fact that PFCs have been used since the 1950s, the concentrations of most PFCs were similar among the four age groups (Table 2, Figures 1 and 2), suggesting that a trend of increasing concentrations with age does not exist. In contrast, concentrations of other persistent pollutants (e.g., organochlorine compounds) in these same NHANES 2001−2002 pools consistently increased with age (Needham LL, personal communication). Previous studies of different populations also reported an increased in concentration with age for persistent organochlorine chemicals (*48*, *49*).

Our findings are consistent with reports showing similar concentrations of PFOS and PFOA, regardless of age (*17*−*19*). PFCs, unlike dioxins and other organochlorine compounds, do not concentrate in adipose tissue. Instead, PFOA and other PFCs have a high affinity for proteins, distribute predominantly to the liver and plasma, and undergo enterohepatic circulation (*32*). PFCs are persistent chemicals with human serum elimination half-lives of ∼4 years for PFOA, 5 years for PFOS, and longer (∼8 years) for PFHxS (Olsen GW, unpublished results). Extensive enterohepatic circulation of PFCs could increase their biological half-lives and may explain the observed lack of a general trend of increasing PFCs concentrations with age.

Interestingly, non-Hispanic white adolescents (12−19 years of age) appeared to have greater concentrations of PFHxS and Me-PFOSA-AcOH than did non-Hispanic white adults 20 years of age and older (Figure 1). Greater mean concentrations of PFHxS and Me-PFOSA-AcOH in children than in adults have been previously reported (*19*). Me-PFOSA-AcOH is a known oxidation product of 2-(*N*-methyl-per-

fluorooctane sulfonamido) ethanol, a compound that was primarily used in surface treatment applications (e.g., carpets, textiles). PFHxS was used as a building block for compounds incorporated in fire-fighting foams and specific postmarket carpet treatment applications. Therefore, one possible explanation for the greater concentrations of these two compounds in these children and adolescents than in adults could be increased exposure due to increased contact with carpeted floors among children and young adults. Furthermore, PFHxS was frequently found in archived house dust samples collected in the United States (Strynar MJ, unpublished results) suggesting that indoor exposure to this compound may be important.

Non-Hispanic white males had greater LSM concentrations of PFOS (40.19 ng/mL) and PFOA (6.98 ng/mL) than Non-Hispanic white females (PFOS, 23.97 ng/mL; PFOA, 3.97 ng/mL). Greater geometric mean concentrations in adult men (PFOS, 37.8 ng/mL; PFOA, 4.9 ng/mL) than in women (PFOS, 32.1 ng/mL; PFOA, 4.2 ng/mL) were also reported in the United States (*17*), and similar trends have been observed in adult populations in Japan (*50*). Non-Hispanic white males also had greater concentrations of PFNA than did Non-Hispanic white females (Figures 1 and 2, Table 3). For Non-Hispanic blacks and Mexican American males and females, the concentrations of all PFCs appeared to be quite comparable (Figures 1 and 2), The reason(s) for these differences are presently unknown.

In a previous study, we found that the prevalence of human exposure to PFCs was very low among a group of Peruvian adults (*11*). We speculated that this was associated with limited use of PFC-containing products (e.g., carpet and textile stain-resistant products, packaging materials for snack foods and fast foods, etc.) in Peru. In contrast, those products are quite prevalent in the United States and other industrialized countries. In the present study, the concentrations of most PFCs differed significantly based on race/ethnicity (Tables 2 and 3). The highest mean concentrations for all PFCs were found among non-Hispanic whites, regardless of sex or age. Mean concentrations were lowest for Mexican Americans. Specifically, non-Hispanic whites had PFCs concentrations about twice those of non-Hispanic blacks, and about 3-fold greater than those of Mexican-Americans. These differences may be a reflection of the greater exposure to PFCs among non-Hispanic whites than among the other racial/ethnic groups examined. Prolonged use of PFCs for various applications, including protective coatings for fabrics and carpet, paper coatings, and insecticide formulations, may be a major contributor to human exposure to these compounds. In addition, genetic variability, diet, lifestyle, or combinations of these factors may contribute to the observed differences.

In summary, this paper provides data on the estimated mean concentrations of PFCs for selected demographic groups in the United States. The high prevalence of exposure to PFCs and differences among race/ethnic groups highlight the need for additional research to identify sources of human exposure to PFCs and to study the environmental distribution of these chemicals. We plan to measure the concentrations of PFCs in a one-third subset of samples collected from NHANES participants, 12 years of age and older, during 1999−2000 and 2003−2004, and in future NHANES. We have conducted similar measurements for other environmental chemicals, including dioxins, PCBs, and organochlorine pesticides, for the years 1999−2000 and 2001−2002 (*51*, *52*). Our data also stress the importance of conducting additional studies to evaluate the potential human health effects resulting from exposures to PFCs.

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